Covid-19: Challenges and solutions – An Overview

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Abstract

Emerging pandemics show that humans are not infallible and communities need to be prepared. Coronavirus outbreak was first reported towards the end of 2019 and has now been declared a pandemic by the World Health Organization. Worldwide countries are responding differently to the virus outbreak. A delay in detection and response has been recorded in China, as well as in other major countries, which led to an overburdening of the local health systems.

On March 11 the World Health Organization officially designated the novel coronavirus outbreak a pandemic. Defined as the worldwide spread of a new disease, such a declaration is the first to be made since the 2009 H1N1 swine flu. As of this writing, there have been approximately 336,000 confirmed cases of the new disease, called COVID-19, resulting in more than 14,600 deaths worldwide. As scientists in the Global Carbon Project, an international group that tracks greenhouse gas emissions, we've been bombarded with questions: How much will carbon dioxide pollution fall this year? Will emissions rise again? Could climate action be the silver lining of COVID?

Real-time estimates of carbon dioxide emissions aren't readily available and often come with a one- or two-year delay. In response, and using new methods we developed, we just published the first study to address daily declines in global carbon dioxide emissions associated with the virus. We gathered data for state, provincial and national economies across six different industries, including transportation, electricity and manufacturing. We combined these data with a confinement index that helped us understand how many billions of people were under shelter-at-home rules and other constraints to slow the spread of the virus.

Keywords— COVID, virus, India, Industries, WHO, Pandemic, European Union, H1N1 swine flu.

Introduction

Whereas COVID-19 is caused by a coronavirus and not an influenza virus, the 1918 flu pandemic—which caused at least 50 million deaths worldwide, according to the Centers for Disease Control and Prevention—might be the best model to understand this novel pathogen's behavior. It is also an outbreak for which massive social interventions were undertaken."Past influenza pandemics give some sense of what the overall [trajectory] of a virus like this would be because the reproductive number of this virus"—defined as how many people each infectious person transmits the disease to in a completely susceptible population—"is pretty similar to that of a pandemic flu," says Marc Lipsitch, a professor of epidemiology and director of the Center for Communicable Disease Dynamics at Harvard University. Although it is difficult to determine exact figures for an emerging disease, reports put the reproductive number of COVID-19 between 2 and 2.5. The median reproductive number for the 1918 flu pandemic was around 1.8. Lipsitch estimates that between

about 20 and 60 percent of the global population will ultimately become infected with the novel coronavirus, or SARS-CoV-2.

Although every virus and resulting disease is different, a look at epidemic dynamics of both COVID-19 and the 1918 flu points to similar successful containment procedures. In a 2007 study published in JAMA, Howard Markel of the Center for the History of Medicine at the University of Michigan Medical School and his co-authors analyzed the excess deaths from pneumonia and influenza (meaning how many more there were than usual during no pandemic years) in 43 U.S. cities from September 8, 1918 through February 22, 1919. Despite the fact that all of the cities implemented no pharmaceutical interventions, it was the timing of activation, the duration and the combination of measures that determined their success. The researchers found "a strong association between early, sustained, and layered application of [such] interventions and mitigating the consequences of the 1918–1919 influenza pandemic in the United States."

COVID-19 has left city squares abandoned and our streets empty. Unemployment in the United States has already topped 15 percent, and the European Union's economy is projected to shrink by more than 7 percent in 2020. But hidden in the fallout from the virus are lessons to make the world a better place. Reducing pollution and reimagining transportation are strong places to start.

This review will help the readers to understand the difference in response by different countries and their outcomes. Based on the experience of these countries, India responded to the pandemic accordingly. Only time will tell how well India has faced the outbreak. We also suggest the future directions that the global community should take to manage and mitigate the emergency. Although a coronavirus—a family of viruses that cause illnesses ranging from the common cold to severe acute respiratory syndrome (SARS)—had not previously triggered a pandemic, this is not the first time we have seen the global transmission of a serious disease. Studying past outbreaks can help scientists better estimate the trajectory of COVID-19 and identify the best measures to slow its spread.

On the other hand, some other nations have put in place effective strategies to contain the infection and have recorded a very low number of cases since the beginning of the pandemics. Restrictive measures like social distancing, lockdown, case detection, isolation, contact tracing, and quarantine of exposed had revealed the most efficient actions to control the disease spreading.

Objective:

This paper intends to study the COVID-19 could permanently transform World the challenges and opportunities

Challenges posed by COVID19 to India and world

For countries under maximum lockdown in April, surface transport activities decreased by half, aviation by three quarters, and power generation by as much as 15 percent, despite small increases in residential use where people stayed home. In total, we found that daily carbon dioxide emissions declined about 17 percent globally at the peak of confinement in early April. Decreases for single countries were temporarily even bigger, with a short-term drop of 32 percent in the U.S. at the low point.

Previous recessions and global crises have had surprisingly little effect on the growth of fossil fuel emissions since World War II. Our data show that the global financial crisis that began in 2008 caused global carbon dioxide emissions to fall 1.4 percent. Emissions roared back in 2010, climbing 5.1 percent, as if nothing had changed—because it hadn't, at least in terms of transforming energy infrastructure and demand. The Great Recession put people out of work and shattered lives. Governments responded with stimulus funding, and when people went back to work, emissions returned with a bang.

Even as climate scientists, therefore, we can't see this year's drop in emissions as any kind of silver lining. COVID-19 has caused too much pain for that. We can learn lessons from it, though, and transportation is the place to start.The fallout from COVID-19 could change commuting and transportation permanently. No one misses commuting an hour (or two) a day. Telecommuting, even part-time, might be the new normal. Families are biking comfortably on what once were choked streets—even at rush hour, which itself could be a thing of the past. Traffic congestion has vaporized. And from India to Indiana, our skies are blue. Pollution from cars and coal still kills millions of people worldwide every year, even more than the incomprehensible losses from the virus so far. Clean energy coupled with electric cars could give us blue skies every day and safer lives—without sheltering in place.

Will COVID-19 create sustained change? It already has. This once-in-a-generation event will shape our psyches permanently. Will it create sustained change in fossil fuel emissions and other air pollution? Not unless we respond by reshaping commuting and transportation around the world. Like a car speeding by us on a freeway, the virus will pass.

The parallel to COVID-19 is obvious. Emissions declines driven by crises are fleeting, typically lasting only a year or two (unless we revisit the Great Depression of the 1930s, which no one wants to do). Only structural changes to our economies can make them permanent.

A drop of 4–7 percent in CO2 emissions for the year, as we project, is the level of reduction needed to keep the world on track to limit climate change well below 2 degrees Celsius. However, this reduction is only for one year. Last fall the U.N. Environment Program suggested emissions needed to drop 7.6 percent every year until 2030 for global temperature increase to stay below the safer limit of 1.5 degrees C. The virus illustrates both what's possible and what's close to impossible. Enforced sheltering-in-place rules and massive unemployment are unsustainable and undesirable ways to cut emissions.

Such emissions declines are enormous and unprecedented—but won't last. Our annual projection for 2020 is for carbon dioxide emissions to fall 4 to 7 percent depending on how quickly the confinement measures ease and how severe the accompanying recession will be. Countries across Asia and Europe, and some U.S. states, are already starting to open their doors despite the lingering presence of the virus. Emissions will start inching back to normal when confinement relaxes.

The most effective class of nonpharmaceutical control measures were those related to social distancing: canceling public gatherings, closing places of worship, schools, bars and restaurants, isolating the sick and quarantining those they came in contact with. (Many cities around the world have adopted such measures in the current outbreak.) "In my opinion, that is probably the most important single class of things to do, as quickly as possible, to slow the spread" of a pandemic,

Lipsitch says. "Waiting until you can see that you have a problem is waiting too long, because there's a delay in seeing the fruits of the measures."

NEW OPERATIONS IN HEATHCARE SECTOR

By undertaking these steps early, populations can also prevent peak demands on their health care systems and flatten the pandemic curve—that is, have a gradual increase in cases over time rather than many all at once. This slowdown is especially important because it can take two or three weeks before those infected with SARS-CoV-2 are sick enough to require intensive care, so demand could spike quickly. In a 2007 Proceedings of the National Academy of Sciences USA paper, Lipsitch and two other researchers showed that during the 1918 influenza pandemic, cities that intervened early and intensively to slow transmission through social distancing, such as such as St. Louis, Mo., had slower epidemics with smaller peaks, compared with those that waited longer to act, such as Philadelphia.

Similarly, in a preprint report, Lipsitch and his colleagues analyzed the timing of control measures and of community spread of COVID-19 in the Chinese cities of Wuhan and Guangzhou from January 10 to February 29, 2020. Wuhan implemented measures such as strict social distancing and quarantining contacts of infected individuals six weeks after sustained local transmission was observed, whereas Guangzhou implemented these measures within one week. The researchers found that early intervention, relative to the course of the disease in the population, resulted in Guangzhou having "lower epidemic sizes and peaks" than Wuhan in the first wave of the outbreak.

Intense public measures are also one reason SARS, which resulted in around 8,000 cases with a global case fatality rate of 11 percent, was eliminated from the population. One difference, however, is that with SARS, those who were infected were likely quite sick before they became very infectious, whereas with COVID-19, people appear to be fairly infectious when they first start developing symptoms—or even before then—according to Lipsitch. In fact, in a paper published last week in Science, researchers note that with the novel coronavirus, "undocumented infections often experience mild, limited or no symptoms and hence go unrecognized, and, depending on their contagiousness and numbers, can expose a far greater portion of the population to virus than would otherwise occur." So despite the lower fatality rate, COVID-19 has resulted in more deaths than SARS and Middle East respiratory syndrome (MERS)—which has a 34 percent case fatality rate—combined.

Other disease countermeasures include making buildings less favorable to viral transmission by humidifying and ventilating them and implementing ongoing communication with the public so it can understand and react appropriately. One issue during the SARS outbreak was that, for a number of months, the government in China actively denied the existence of the disease. Instead people relied on text messages and rumors about a new killer flu.

"Because the government wasn't proving itself to be reliable, it became that much harder to actually address the outbreak. And it allowed the disease to really take more of a hold than it might otherwise have," Duluth's Youde says.

In order to slow down epidemics and pandemics, either the conditions for transmission need to become unfavorable over a long period of time or enough people have to become immune so that transmission cannot pick up again if the virus is reintroduced. The latter scenario, of course, means the fraction of the population that is immune has to be high enough so that each contact and infected case creates fewer than a single new one.

Regular flu and cold viruses have a strongly seasonal pattern of infectiousness in temperate regions such as the continental U.S. This seasonality is partly related to changing weather conditions and how easily the pathogens are transmitted, but it is also because of the number of susceptible hosts as people are made immune by past exposure. The same is not true of new viruses, such as the one that causes COVID-19, however.

INDUSTRIAL AND MEDICAL SOLUTIONS TO FIGHT PANDEMIC

"Pandemics happen out of season. And pandemic viruses have the whole world before them," says Lipsitch, who explains that the advantage for novel viruses is that almost no one is immune to them. Seasonal viruses, on the other hand, operate on a thinner margin—meaning the majority of people have some immunity. So those pathogens are most successful when conditions for transmission are most favorable, which is usually winter. With COVID-19, Lipsitch adds, "I think [it's] more likely seasonal changes will modestly reduce the rate of transmission and maybe slow things down—but probably not to the point of making the number of cases [decrease but rather] go up more slowly."

For now, a coordinated global effort among researchers, countries, and nongovernmental and international organizations is necessary to address the current pandemic itself while learning basic information about the virus and its spread dynamics. "In terms of having some sort of international response, we're trying to build the airplane as we're flying it," Youde says.

A virus is an unusual beast. Essentially it is a cluster of genetic material that integrates itself into a cell and takes over some of the cell's molecular machinery, using it to assemble an army of viral copies. Those clones burst out of the cell, destroying it, and go on to infect nearby cells. Viruses are hard to kill off completely because of their cellular integration—they hide within their hosts. And they have explosive reproductive rates. Because total eradication is so hard, antiviral drugs instead aim to limit replication to low levels that cannot hurt the body.

In 2013 Denison and Ralph Baric, a coronavirus researcher at the University of North Carolina at Chapel Hill, identified a vulnerable site on a protein common to all coronaviruses they had examined, a spot that is key to the microbe's ability to make copies of itself. If that ability is hindered, a coronavirus cannot cause widespread infection. Four years later researchers in the two laboratories spotted a compound that acted on this protein site. It was sitting, unused, in a large library of antiviral compounds created by the biotech giant Gilead Biosciences. The scientists got a sample and, in test tube and animal experiments, showed that the drug, called remdesivir, shut down the replicating machinery of several coronavirus variants.

So in early January, when the alarms rang about SARS-CoV-2, Denison and Baric alerted colleagues at Gilead that they were sitting on a potential treatment. Largely because of its activity against other coronavirus strains in Denison and Baric's animal studies, remdesivir was made available to patients for "compassionate use" in January. By March, Gilead had rushed the compound into two human trials, planning to test the drug's safety and most effective doses on about 1,000 ill patients over several months; health authorities in China began two similar trials. While that was happening, Denison, Baric and a group of their colleagues at Emory University identified still another compound, called EIDD-2801, that hits the same viral vulnerability. In early April they published results showing that in mice, the new substance helped breathing and reduced the amount of many coronaviruses. In test-tube experiments with human lung cells, it drastically hindered SARS-CoV-2.

COPY STOPPERS PATENTING VACCINATION

All coronaviruses use the same mechanism to reproduce, which involves an enzyme called viral RNA polymerase, so Baric says that was an obvious target. The polymerase makes lots of mistakes as it copies the virus, and it relies on another enzyme, known as an exonuclease, to "proofread" and fix them. Remdesivir appears to disable the proofreading enzyme. Then the virus's copying factory becomes sloppy and produces fewer new viruses.

EIDD-2801, the compound with promising animal and test-tube results reported in early April, aims at the same viral enzyme. But unlike remdesivir, which much be given intravenously, EIDD-2801 can be taken as a pill. For this reason, Baric and other researchers investigating EIDD-2801, including George Painter, a professor of pharmacology and president of the Emory Institute for Drug Development, which first produced the drug, suspect it may end up being more widely used than remdesivir.

In 2018 Painter and his colleagues identified EIDD-2801's activity during a search for a universal influenza medicine. When SARS-CoV-2 emerged, Painter's group immediately shifted focus. EIDD-2801, like remdesivir, inhibits the coronavirus's self-copying operations, but it also works against virus variants with a mutation that made them resistant to the Gilead drug. In addition, EIDD-2801 is effective against a host of other RNA viruses, so it could serve as a multipurpose antiviral, much as some antibiotics can work against a wide variety of bacteria. For COVID-19, says Wayne Holman, co-founder of Miami-based Ridgeback Biotherapeutics, which has licensed the drug and is planning clinical trials, the goal is to have a pill that can be taken by patients at home early in the course of the disease to prevent it from progressing.

BLOCKING INFECTION THE NEW OPPORTUNITIES

To stop SARS-CoV-2 from penetrating cells in the first place, scientists are trying to develop antibodies that lock onto the viral protein that facilitates cell entry, a part of the virus known as the spike. Some of these neutralizing antibodies, made of a protein called immunoglobulin, may come from the blood of patients who have already cleared the virus. Several medical centers, including Johns Hopkins Hospital and the Mayo Clinic, are harvesting blood plasma from

survivors and screening it for antibodies. In a technique known as convalescent therapy, doctors then transfuse it into hospitalized patients with life-threatening acute respiratory distress. Early studies of a few such patients suggest the approach may work—some patients' symptoms improved, and levels of the virus in their bodies dropped—but the work is very preliminary.

Takeda Pharmaceuticals, a Japanese firm, is also collecting plasma from recovered COVID-19 patients to identify antibodies. In that plasma, the company is identifying antibodies that show the most activity against SARS-CoV-2. Using these antibodies as a template, the Takeda researchers plan to synthesize a batch of even more active versions to create a potent cocktail of infection inhibitors, says Chris Morabito, head of research and development of plasma-derived therapies. The therapy—TAK-888—might enter clinical trials by year's end, Morabito says; the number "888" represents "triple fortune" in Chinese. Several other drugmakers, including Regeneron and Vir Biotechnology, are generating their own therapeutic antibodies and say they will also be tested in patients this year.

Another blockade strategy focuses on the cellular docking site that the virus uses. Josef Penninger, a molecular biologist at the University of British Columbia in Vancouver and founder of drug company Apeiron Biologics, is trying to lure the virus away from a chemical receptor called ACE2 in the outer wall of lung cells. The coronavirus spike protein binds to this receptor. Several years ago Penninger's lab synthesized a decoy version of ACE2. In test-tube experiments, the scientists found the synthetic molecule—APN01—attracted coronaviruses away from real human airway cells. The virus locked onto the decoy and was marooned there. "We are blocking the door for the virus and, at the same time, protecting tissues," Penninger says. Apeiron is planning clinical trials later this year for APN01, which must be administered in the hospital as an infusion to sick patients.

Conclusion

Labs around the world, like Denison's and Baric's, have logged years of experience poking about the inner workings of coronaviruses because of SARS and MERS. By the time the new coronavirus was genetically sequenced and its structure revealed, scientists already had identified the enzymes and proteins that most coronaviruses use to spread from one infected human cell to another and also understood that the body could create an overly aggressive inflammatory response when the virus infected lung airway cells.

Because of this work, three main strategies for impeding the virus have emerged as the labs have turned to the current threat. One strategy is to find compounds like remdesivir and EIDD-2801 that gum up the virus's reproductive machinery when it enters a target cell. A second is to block the virus, like a bouncer outside a bar, from entering and infecting those cells in the first place. The third approach is to muffle the immune system's dangerously overactive response, a "cytokine storm" that can drown a victim in a mass of congestion and dying airway cells.

To find these drugs, researchers have turned to the Food and Drug Administration's list of some 20,000 compounds approved for human use and crawled through drug patent applications looking for compounds with promising mechanisms of action. The goal has been to find drugs that have been at least partly developed, avoiding years of making therapeutic molecules from scratch. The Milken Institute, a health advocacy think tank, counted 133 experimental COVID-19 treatments in mid-April. About 49 of these therapies are being rushed into clinical trials. Their effectiveness in people is not yet known, and scientists caution that such drugs, like other antivirals, are unlikely to be cures. But they could reduce symptoms enough to give patients' immune systems a chance to beat the virus on their own.

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